



REVISTA BRASILEIRA DE ANESTESIOLOGIA

Publicação Oficial da Sociedade Brasileira de Anestesiologia
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CLINICAL INFORMATION

Spinal subarachnoid haematoma after spinal anaesthesia: case report



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Received 9 March 2015; accepted 17 August 2015

Available online 21 January 2016

KEYWORDS

Subarachnoid
haematoma;
Spinal anaesthesia;
Risk factors

Abstract Subarachnoid haematoma after spinal anaesthesia is known to be very rare. In the majority of these cases, spinal anaesthesia was difficult to perform and/or unsuccessful; other risk factors included antiplatelet or anticoagulation therapy, and direct spinal cord trauma. We report a case of subarachnoid haematoma after spinal anaesthesia in a young patient without risk factors.

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PALAVRAS-CHAVE

Hematoma
subaracnoideo;
Raquianestesia;
Fatores de risco

Hematoma espinhal subaracnoideo após raquianestesia: relato de caso

Resumo Hematoma subaracnoideo após anestesia espinal é conhecido por ser muito raro. Na maior desses casos, a anestesia espinal foi difícil de executar e/ou mal sucedida; outros fatores de risco incluem terapia anticoagulante ou antiplaquetária e trauma medular direto. Relatamos um caso de hematoma subaracnoideo após raquianestesia em paciente jovem sem fatores de risco.

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Introduction

Subarachnoid haematoma after spinal anaesthesia is known to be very rare.^{1–3} In the majority of these cases, spinal anaesthesia was difficult to perform and/or unsuccessful;

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other risk factors included antiplatelet or anticoagulation therapy, and direct spinal cord trauma.^{2,3} We report a case of subarachnoid haematoma after spinal anaesthesia in a young patient without risk factors.

Case report

Male patient, 32 years old, 72 kg, 180 cm, who presented for open inguinal hernia repair. Past surgical history consisted of some minor surgeries under local anaesthesia and a nasal operation under general anaesthesia. The patient had a negative past medical history, no history of abnormal clinical bleeding and was not taking any medications. His ASA physical status classification was one.

For this operation, the patient requested spinal anaesthesia at the preoperative anaesthetic consultation. Following French Society of Anaesthesia and Intensive Care recommendation, no pre-operative investigation was requested.⁴

The patient was placed in a sitting position. Spinal anaesthesia was attempted at the L3-4 intervertebral space via a midline approach using a BD™ Whitacre 25G spinal needle with pencil point. At the first attempt, clear cerebrospinal fluid was observed without any particular difficulties.

Hyperbaric bupivacaine 0.5% 2.0 mL was injected. Intrathecal needle tip placement was confirmed prior to, during and after injection by free aspiration of cerebrospinal fluid. No incidents such as blood or paraesthesia were reported. Loss of sensation to cold touch was used to determine the dermatome level of the block which was T8 bilaterally.

The surgery began 10 min after spinal anaesthesia was performed and lasted 20 min without any problems. Intra operatively, a total of 80 mg of propofol was given for sedation, as well as 2 g of cefazolin for antimicrobial prophylaxis, 8 mg of dexamethasone, 100 mg of ketoprofen, 20 mg of nefopam and 1 g of acetaminophen.

The patient remained in the post-anaesthesia care unit for one hour and then returned to the ward. The Modified Aldrete Scoring System was 10. No prophylaxis was given for deep vein thrombosis. He was discharged from the hospital in the afternoon and the post-anaesthetic discharge scoring system was 9.

On the 9th postoperative day he presented to the Emergency Department complaining of intense lower back pain for 3 days, radiating to both legs following L5-S1 sensitive territory associated with paresthesia. These symptoms began with hypoesthesia of the perineum on the first postoperative day and gradually worsened leaving him unable to walk. Further investigation by the neurologist revealed that the sensory block lasted more than 24 h after discharge.

Neurological examination revealed no loss of power, sensation or sphincter control but loss of reflexes in the lower limbs. The pain was reproducible on clinical examination and needed level 3 analgesic drugs to be soothed. Since cauda equina syndrome was suspected a magnetic resonance imaging (MRI) was performed. It revealed a 35 mm × 8.5 mm subarachnoid haematoma at level L4-L5 associated with a small epidural haematoma at the cauda equina with no damage to the filum (Fig. 1). The lumbar canal was large and there was no meninges abnormality. There was no



Figure 1 T1 weighted sagittal MRI showing subarachnoid haematoma regarding L4-L5 associated with a small epidural haematoma regarding the cauda equina with no damage to the filum.

abnormality in the coagulation profile and no inflammatory syndrome. Subarachnoid haematoma diagnosis was retained and a neurosurgeon was consulted. No surgical intervention was needed so the patient was hospitalized for 3 days in neurology for conservative treatment and pain management. He was then discharged with pain treatment consisting of opioids, nonsteroidal anti-inflammatory and pregabalin. Follow-up at one month revealed residual lower back pain with radiation to the knees, walking lameness and stiff lower back. There was no more loss of lower limbs reflexes. An MRI control scan revealed no signs left of the initial haematoma and no nerve damage.

Discussion

Spinal subarachnoid haematoma is a very rare complication of spinal anaesthesia.⁵ Spinal subarachnoid haematoma cases reported up to now were due to difficult and unsuccessful spinal anaesthesia. Several predisposing factors have been identified such as problems with blood coagulability, antiplatelet and anticoagulant therapy, direct spinal cord trauma, spinal stenosis, combined spinal-epidural anaesthesia, difficult traumatic and multiple punctures. Antiplatelet or anticoagulation therapy is a major role factor in subarachnoid bleeding, and can be avoided if guidelines for safe practice are followed.^{5,6} However, sometimes etiological factor that explained that bleeding could not be identified.¹

Spinal and epidural anaesthetic procedures in combination with anticoagulant therapy represent the fifth most common etiological group and spinal and epidural anaesthetic procedures alone represent the tenth most common cause of spinal haematoma.¹ Idiopathic spinal haematoma, cases related to anticoagulant therapy and vascular

malformations represent the first, second and third most common categories respectively.¹

In a Finland study incidence of neuraxial haematoma after spinal block was 1:775,000.² They reported one case of subarachnoid haematoma after spinal block. A 67 year old woman admitted for arthroscopy. Spinal stenosis might have contributed to the neurologic symptoms for the latter case. Small space due to herniation, arachnoidites, spondylo atrodic process, or thickening of the ligamentum flavum leading to lower circulation of cerebrospinal fluid and contributing to the formation of subarachnoid haematoma.

Bleeding in the subdural space could be also related to puncture of the radiculomedullary vessels found along the nerve roots and may be punctured especially if the point of the needle is not in the mid line.⁷ Other predisposing factors include the number of punctures, multiple degenerative discopathy, and spinal stenosis.⁸⁻¹¹ The case we report is highly unusual since subarachnoid haematoma is very uncommon in patients as young as 32 years old.¹² In our case, all preoperative recommendations were followed. No contributing factor has been identified and no incidents such as blood or paraesthesia were reported. No spinal stenosis was spotted on the MRI; only an atraumatic single mid-line puncture with a 25G Whitcare needle was performed. No antiplatelet, anticoagulation or coagulopathy disorders were involved.

However, probably needle size and shape could influence spinal subarachnoid haematoma because fine needles (27G and 29G), may have less vessels traumatises along the length of nerve roots.^{13,14} Some authors suggest that subarachnoid haematoma occurs when radicular vessels are lacerated by traumatic lumbar puncture.^{13,14} Blood in the subarachnoid space usually does not clot, probably because of the great dilution by spinal fluid. Defibrination of blood from the pulsatile motion derived from the brain and spinal cord may be an additional factor.^{11,13,14}

Typical symptoms of subarachnoid haematoma include: spinal root pain, lumbalgia, paraparesis, sphincter dysfunction and headache that does not fulfil the criteria of postdural puncture headache (PDPH). Clinical presentation of intraspinal haematoma may vary from persistent back pain to frank paraplegia.¹⁵ Recommended treatment for symptomatic compression of nerves is emergency spinal laminectomy within 6h. But absence of clinical signs of compression should lead to medical treatment in accordance with neurosurgeon approval.

Early diagnosis is the key to full recovery of subarachnoid haematoma. When suspected, according to the history of spinal anaesthesia and typical symptoms, an emergency MRI should be performed. Computed tomographic scanner does not give conclusive results; only MRI can confirm the diagnosis of subarachnoid haematoma, its localization, size and compression of the filum, if present. It also detects vascular lesions or associated malformation.

A headache following an intradural puncture should lead to suspicion of differential diagnosis including PDPH, drug related headache, intracranial hypertension, meningitis, thrombosis of intracranial veins, cerebral abscess,

subdural or subarachnoid haemorrhage as well as subarachnoid haematoma even if rare, should be borne in mind.

Taking into account all predisposing factors of spinal subarachnoid haematoma after spinal anaesthesia, none were found in our case. The probability of having such a case is extremely rare but possible even if guidelines for safe practice are followed. Any clinical suspicion about such a case should lead to MRI immediately in order to confirm diagnosis and treat before severe permanent neurological damage occurs.

Conflicts of interest

The authors declare no conflicts of interest.

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